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## Relationship of PTEN mutations and EGFR amplification with p27 and cyclin D1 through Akt in glioblastomas

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title of abstract: RELATIONSHIP OF OTEN MUTATIONS AND EGFR AMPLIFICATION  
WITH p27 AND CYCLIN D1 THROUGH Akt IN GLIOBLASTOMAS

authors on abstract: Davide SCHIFFER, Valentina FIANO, Chiara GHIMENTI  
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abstract: Downstream PIP3 generation by PI-3 Kinase activates Akt which inactivates  
AFX/FKHR with the consequent decrease of p27/Kip.1 expression and enhancement of  
cyclin D1 expression. PTEN lipid phosphatase degrades PIP3 and negatively regulates Akt,  
whereas its loss abrogates the negative regulation of Akt which can thus suppress pro-  
apoptotic function of BAD and caspase-9. The same pathway can be followed by activation of  
PI-3 Kinase by EGFR. p27/Kip.1 has been certainly found down-regulated by deltaEGFR. In  
glioblastomas, especially in primary ones, PTEN is mutated in 27-40% of cases and EGFR  
amplified in 60-65% of cases.

PTEN mutations and EGFR amplification by PCR, Akt, p27/Kip.1 and cyclin D1 by  
immunohistochemistry with relevant antibodies and immunoblotting, apoptosis by TUNEL  
and LI of Ki.67 MIB.1 were studied in a series of 75 operated glioblastomas and compared  
among them and with survival. EGFR amplification and PTEN mutations were present in  
40% and 30% respectively of glioblastomas and simultaneously in 7 cases. A relationship  
between EGFR amplification and PTEN mutations, evaluated separately, and p27/Kip.1 and  
cyclin D1 was not clearly found, not even in cases with both alterations together. For Akt we  
could not obtain till now reliable results. p27/Kip.1 and cyclin D1 are maybe also under other  
regulatory mechanisms.

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